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Reply to the Editor:

We also recognize the importance of maintaining the integrity of the clavicle with its articulation with the manubrium and its muscular insertions. Spaggiari and Pastorino concluded that our method does not maintain the integrity of the clavicle because there was insufficient fixation in 3 of our 5 cases. However, they were the early cases. Our problem in the early period lay in the method of fixing the sectioned clavicle. However, besides gaining some technical experience, we solved the problem by fixing the clavicle with a new material, slightly thicker titanium plates. No problems at all have been encountered in the 2 most recent cases. The patients are even bowling with the arm on the side of the operation.

Section of the manubrium is also possible with our method. When performed, it would probably be done by the ordinary thoracic approach. Spaggiari and Pastorino have stated that our method and their method are completely different procedures. Although we also considered the procedure from a totally different viewpoint, when we evaluated the matter thoroughly, it seemed reasonable to conclude that our method is also a TMA.

The main portion of our report says that "sectioning the clavicle in the TMA makes it possible to enlarge the field of vision and at the same time maintain clavicle integrity." An approach to a fairly large portion of the great vessels in the thoracocervical area may very well be possible without sectioning the clavicle if the procedure is performed carefully. However, the field of vision obtained by sectioning the clavicle makes it possible to visualize the great vessels in the thoracocervical area frontally in a way that cannot be compared with the exposure available when the clavicle is not sectioned. The field of vision must be enlarged if the operation is to be performed safely.

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Homograft valve failure in children

To the Editor:

We read with interest the article by Rajani, Mee, and Ratliff.[1] Their finding of a lymphocytic infiltrate associated with rapid failure of cryopreserved homografts in infants lends support to the hypothesis of an immunologic injury being at least partially responsible for this failure. This concurs with our own clinical[2] and laboratory[3] findings but is not supported by others.[4]

Rajani, Mee, and Ratliff end their article with speculation about the possible use of tissue matching and immunosuppression in these patients. Although we believe that there is evidence for an immunologic component to this accelerated failure, we would caution against the empiric use of immunosuppression in these children without further controlled studies. In a small pilot study we transplanted cryopreserved aortic valve grafts into 21 outbred rats as previously described[3] and randomly assigned 11 to receive oral cyclosporine (10 mg/kg per day) (INN: ciclosporin) for 56 days while the remaining 10 received a placebo. The striking finding of this study was that only 3 of the 11 grafts in the cyclosporine-treated group remained patent at 8 weeks whereas 8 of 10 in the placebo group were patent ($P = .03$, Fisher's exact test). The patent cyclosporine grafts did have less medial necrosis and perivascular inflammation than control grafts. The cause of this high occlusion rate in the cyclosporine-treated group is unknown but may be due to a direct toxic effect of cyclosporine on the endothelium of these small grafts.[5] Whether the same effect would occur in larger grafts is unknown.

It is likely that these avascular cryopreserved grafts fail by mechanisms of rejection that are substantially different from those affecting vascularized whole organs. Thus directly transferring immunosuppressive regimens used successfully for cardiac or renal transplantation may not be appropriate. We postulate that there is an immune-mediated attack on these grafts that damages their structural elements, making them more susceptible to early degeneration. Much more work is required to determine if this indeed is the case and to determine how best to minimize this response. We do not believe that sufficient data are yet available to recommend immunosuppression in children requiring homograft valves.

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[Response declined]

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Effect of normoxic cardiopulmonary bypass on leukocyte elastase release

To the Editor:

We read with great interest the recent paper by Ihnken and coworkers[1] about the reduction of oxygen-derived free radicals and nitric oxide by normoxic cardiopulmonary bypass (CPB). In their article, the authors showed that normoxic CPB reduced release of neutrophil elastase in patients undergoing cardiac operations. They wrote, "this is the first time that a Po_2-dependent elastase release on CPB is described."

Recently, we have undertaken a prospective study on 60 patients undergoing cardiac operations to compare the efficiency and safety of different membrane oxygenators. A large variation in Po_2 appeared during perfusion, which gave us an opportunity to assess whether elastase release during CPB was dependent on the level of Po_2. During CPB, arterial Po_2 was measured 5 times: at the start of cooling, during cooling down to 32°C, on stabilized hypothermia at 28°C, during rewarming to 32°C, and at the end of CPB. Elastase was measured before and at the end of CPB from blood samples taken from the radial artery using the same method that Ihnken and associates described (enzyme-linked immunosorbent assay; Merck, Darmstadt, Germany). Results showed that Po_2 varied between 107 and 440 mm Hg at the start of cooling and between 80 and 308 mm Hg on stabilized hypothermia during CPB. On an average of the 5 time points during CPB, Po_2 varied between 128 and 317 mm Hg. Elastase increased in the majority of patients at the end of CPB with a concentration ranging between 41 to 490 ng/mL, or a percentage increase (release) of 47% to 742% compared with baseline concentration before CPB. However, there was no correlation either between Po_2 and elastase concentration at the end of CPB or between Po_2 and the percentage increase compared with the baseline elastase (Fig 1).

Our results suggest that systemic elastase release during CPB is not dependent on Po_2. For years, elastase release during CPB has been known to be largely attributed to blood interaction with the artificial surface of the extracorporeal circuit.[2,3] Modification of the material surface of the circuit has been associated with a reduction in elastase release either during clinical CPB[4] or in a simulated model of CPB.[5] It could well be that normoxic CPB had reduced the cardiac source of elastase but that the effect had been systematically counteracted by other factors, such as blood-material interaction. Thus whether Po_2 plays a role in controlling local or systemic elastase release is of interest, but needs to be confirmed by later studies as normoxic CPB becomes more prevalent than hyperoxic CPB.

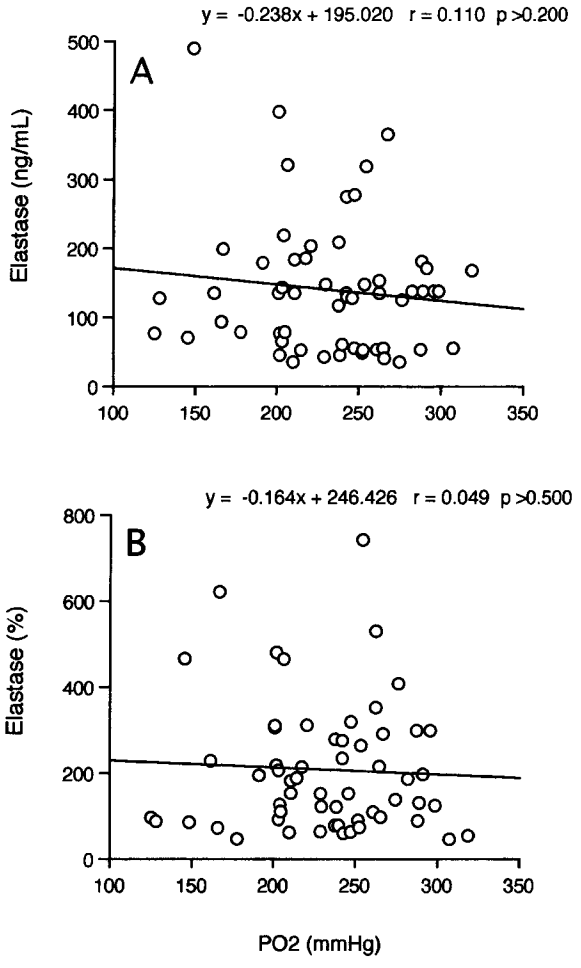

Fig 1. Scattergrams showing no relationship between arterial Po_2 and systemic elastase concentration determined at the end of CPB **(A)** or the percentage increase at the end of CPB compared with the baseline elastase **(B).** Po_2 is the average of 5 blood gas samples determined during CPB (see text for details).

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